

UNIVERSITY OF TORONTO MEDICAL JOURNAL



RESPIRATORY SYSTEM

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VOLUME XXXIV

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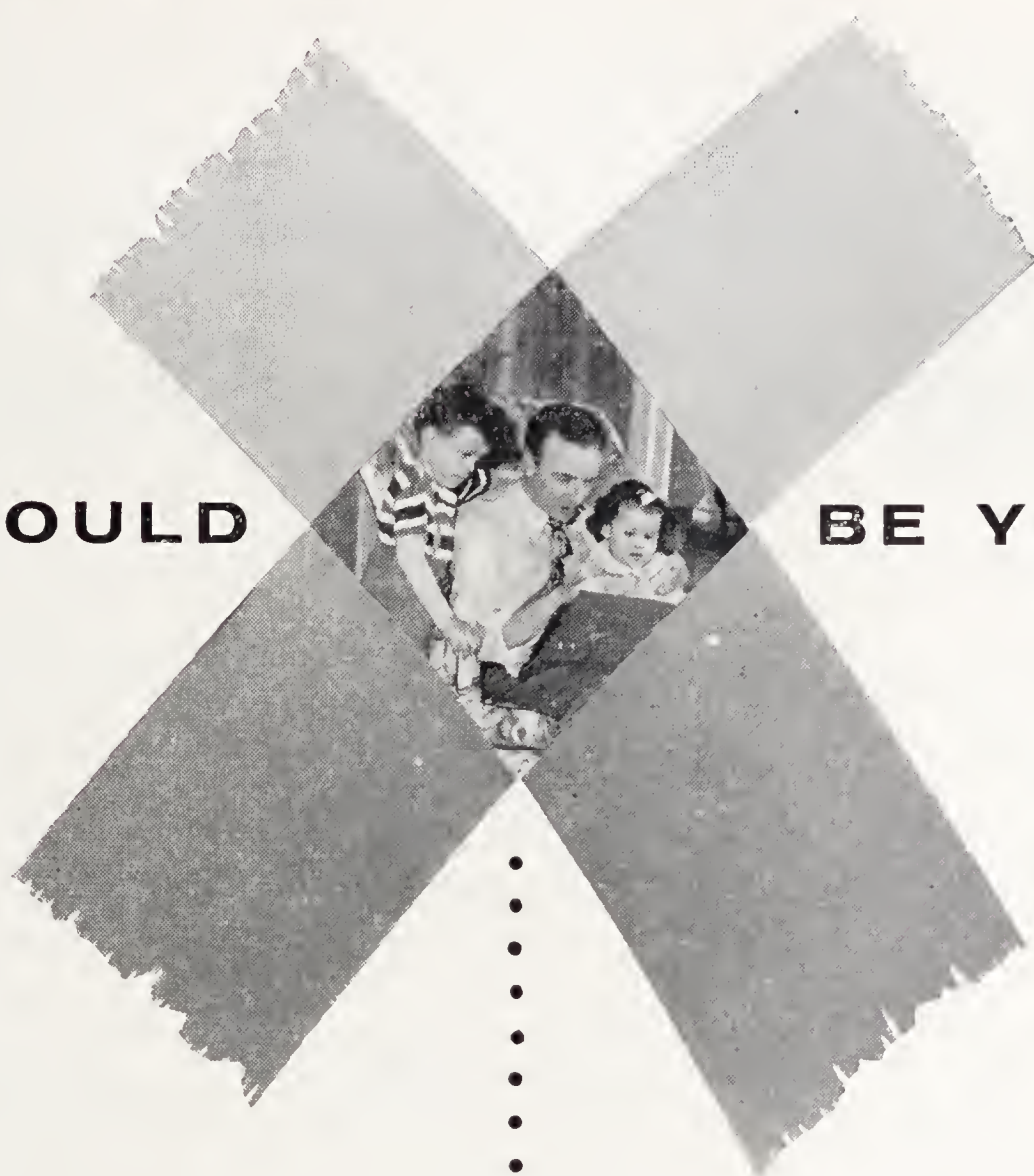
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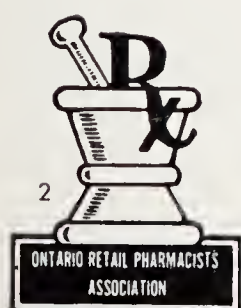
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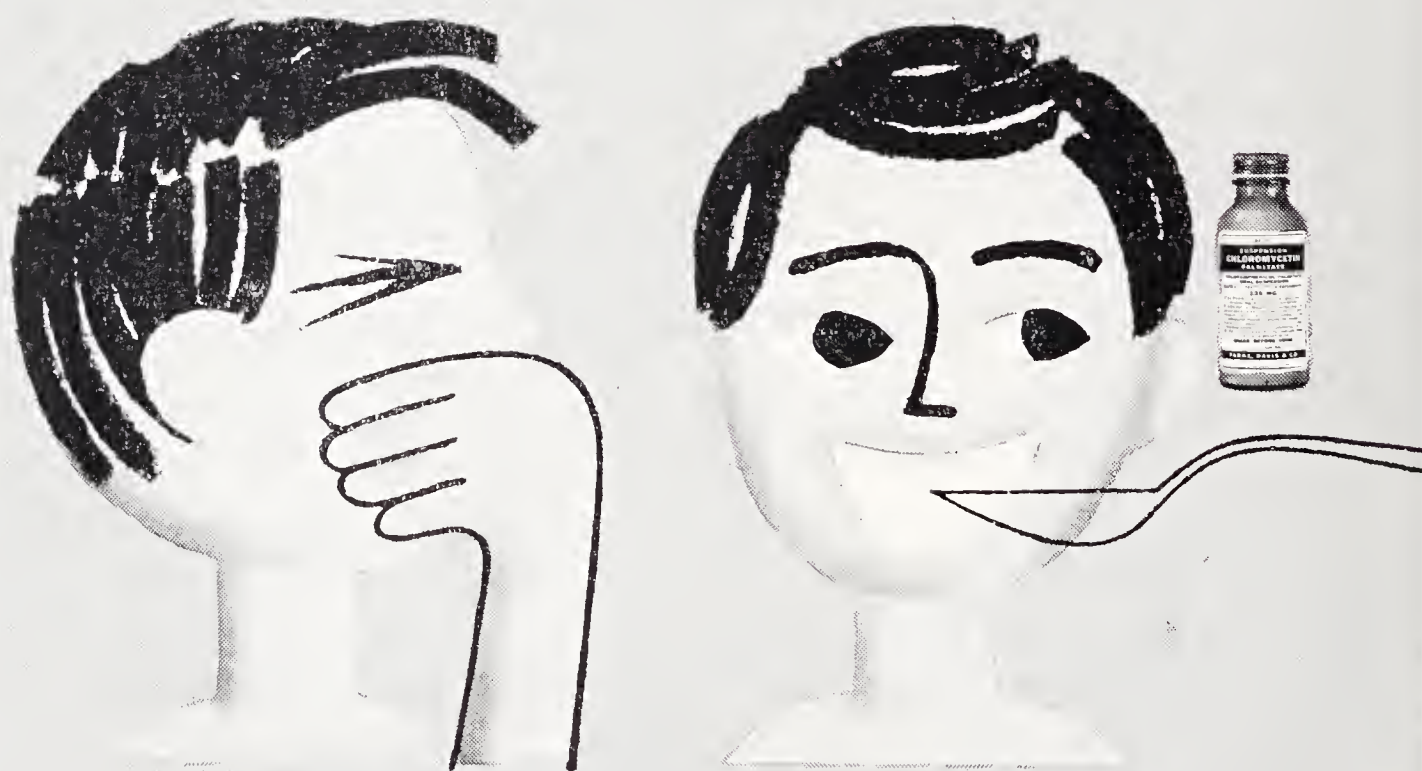
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EDITORIALS

INTERNESHIP—THE DECISION

The problem of selecting a junior internship which has preoccupied the fourth year student during the past few months has aroused the usual amount of speculation about future changes and restrictions, adverse comments and criticism. These comments are directed to a wide range of subjects from the size of the honorarium to the share of responsibility each interne receives. The underlying feeling which motivates many of these comments is usually the student's idea that as an interne, he is not going to be regarded as an individual, but is still looked upon as a "student", a part of a group who as individuals, know very little, do things wrong, are capable of performing only the more menial jobs and are regarded merely as members of the mass. This may partly explain the criticisms directed at more specific aspects of the interne year.

More important however, it is a manifestation of the stress imposed on the student by the change that is about to occur in his life. This is the transition from the life of the "student-doctor", where nothing is irrevocable and the only purpose of performing is for practice, to the life of the fully-fledged doctor, where every decision affects someone else and he must accept the consequences of decisions whether he likes them or not. In other words it is the transition between having responsibility only to himself and having a more serious responsibility to many other people. Internship is the first step in the transition. This transition imposes more stress on each of us than we commonly realize and many of us release the pressure in criticism and comment.

On the other hand it is probably true that in the larger hospitals, which have a relatively large senior staff, the average junior interne does not get as much responsibility as he would like, and which is required ideally for proper sharpening of his observative and deductive faculties. Unfortunately this criticism can be overcome only to a limited extent, and it is also modified by the initiative of the interne himself. But this, nevertheless, is one of the factors which he must consider.

From time to time rumours are circulated that our choice is to be restricted to a limited group of hospitals or restrictions are to be imposed on the type of internship we may do. These rumours usually turn out to be ill-founded, and it may be added that imposing such restrictions would do nothing to further the teaching standards in this country. On the other side we have heard it said by more senior members of the profession that too many of our graduates, in whom this country has an investment, leave this country to interne. These graduates are therefore classed as being "disloyal". One factor is of course higher salaries, but this does not explain the whole situation. They go because they feel that the sum-total of teaching, salary, and responsibility are better than in a comparable hospital here. Imposing restrictions against those few who do go abroad is not the answer to the problem of developing better teaching centers here; it just

makes those who do go less likely to return. And rumours of this kind which are hard to confirm or deny, do not encourage the best relations with a student already very sensitive to any suspected curtailment to his freedom of action. We have heard no definite statement on these matters from anyone who would be likely to know and we hope that they will continue to be merely rumours.

POLICY AND CULTURE

This issue continues the policy of having one issue devoted to one topic. We also include an article of more general interest, a biography of a famous French physician. It has been suggested by several friends of the Journal, that we present more non-medical items, and things of a more cultural nature. We have decided that the sayings of Osler and Harvey, chopped from context and used merely to fill a page end, do not answer this need. Now all we require is some material that does.

ASTHMA — SOME FEATURES OF DIAGNOSIS AND TREATMENT

J. D. L. FITZGERALD, M.D.

ASTHMA, or wheezing due to partial obstruction of the bronchial tree, can be produced in several ways. Mechanical obstruction due to a tumour may cause localized wheezing in one part of the chest. Left ventricular heart failure may cause wheezing under certain circumstances. Chronic infection of the bronchi is frequently associated with coughing and wheezing. For the present, however, we will discuss asthma produced by allergic inflammation of the bronchial mucosa due to antigen-antibody reactions, although it is not always possible to prove the participation of an immune mechanism in all such cases of asthma.

There are two large groups of patients with asthma. First, those with a positive family or personal history of previous allergic disease, more often present in people under 40, with an easily identifiable allergic mechanism. Diagnosis is usually simple, and treatment very satisfactory. The second group of patients have asthma often starting after 40 years of age, frequently without obvious family or personal histories of allergy and without an identifiable allergic mechanism in the pathogenesis. Bacterial allergy, following respiratory infection is thought to be a common factor in this group. Diagnosis is more difficult, and treatment less satisfactory than in the first group. These two groups have been widely described as *extrinsic* asthma and *intrinsic* asthma, respectively. Unfortunately, a mixture of both types is often present, and these terms become less useful. Careful questioning will often reveal an allergic background in both *extrinsic* and *intrinsic patients*. This fact has directed attention to the inherited

constitutional defect (*atopy*) in patients with asthma and is offered as partial explanation of why these people react with wheezing on exposure to inhaled substances (pollens, danders, dusts), foods, or infection, when other people never develop asthma in similar circumstances. Why asthma develops at all in such predisposed people is still not known. But it is recognized that anyone with this inherited tendency can develop asthma at any time, often reacting to substances that they had contacted previously without symptoms developing.

The variability of onset and severity of asthma has often led to undue importance being attached to factors that frequently "trigger" asthmatic attacks, such as weather changes, fatigue, emotional upsets, etc. Without a basic atopic mechanism being present, such precipitating factors would not produce asthma, but they might just as well cause a flare-up of ulcerative colitis or rheumatoid arthritis in persons with predisposition to those diseases. This is not to discount the importance of these various "trigger" factors in the management of patients with chronic, intractable asthma.

The question of the *asthma personality* should receive some comment. Some physicians feel that asthma develops only in people with a certain psychological make-up, and with certain typical experiences in their early developmental period. Consequently the opportunity to undertake allergic investigation of asthmatic patients is often ignored, on the bases that there is "nothing to be done" for such people. There is very real evidence that contradicts the *asthma personality* point of view. For example the theory of "maternal rejection" as the starting

point for asthma has not been supported by observations made in a large Home for rejected children. The incidence of asthma in this population was actually a little lower than that in the general population. Perhaps the best evidence of the heterogeneity of asthmatics is seen in the clinic, where many people of varying colours, races, and psychobiological backgrounds can be observed. Whether one holds the aetiological views of some psychoanalysts, or the more commonly held concept of atopic inheritance plus non-allergic "trigger" factors in the pathogenesis of asthma, it is imperative that all possible factors be considered in diagnosis and treatment directed accordingly.

DIAGNOSIS:

Anyone who has seen a case of acute asthma will agree that a diagnosis of asthma is not difficult to make. However, it takes considerable time to elucidate the causative and contributing factors, and assess their significance. A searching history combined with physical examination will often reveal the significant points for specific diagnosis. A positive family history of allergic disease, and particularly a positive personal history of earlier allergic reactions, (e.g. atopic dermatitis; allergic rhinitis; food allergy) will confirm the patient's atopic background. The age of onset of symptoms, the frequency of attacks, seasonal variations, or onset following respiratory infections are all helpful clues.

Physical examination of the chest may be negative unless the patient is having an attack. In chronic cases of long duration emphysema may be present. When wheezing is present it is bilateral, diffuse and widespread. Breath sounds may be surprisingly quiet in very severe "status asthmaticus" of long duration, due to obstructive emphysema, with small tidal air volume. In spite of "struggling for air", and using the accessory muscles

of respiration, ventilatory exchange is very small. Because of the relative "quietness" of the chest, the error of underestimating the severity of bronchial obstruction is sometimes made. In less severe asthma, loud rhonchi are usually present, as ventilation is less obstructed.

One of the most telltale findings in patients with asthma is the boggy, pale, bluish-coloured mucous membrane of the nose. This does not have to be associated with acute nasal symptoms, but is often the first physical evidence that the patient belongs in the atopic population.

LABORATORY EXAMINATION:

1. *Blood*—eosinophilia (in the absence of other causes) and relative lymphocytosis are often found in the peripheral blood, almost constantly in asthma associated with infection.

2. *Sputum*—mucoid, tenacious sputum often without any purulent component, is common. The presence of recently purulent sputum is most helpful in recognizing recent infection in an exacerbation of asthma.

3. *X-rays*—sinus films frequently reveal asymptomatic hyperplastic sinusitis. X-ray films or fluoroscopy of the chest may be negative, or may show emphysema, or other evidence of chronic bronchial infection. Fluoroscopy is most helpful in assessing total respiratory function, and also permits observation of the heart size and action.

4. *Allergy tests*—Within limits, special skin tests with a selection of appropriate antigens will often help confirm the clinical impression gained from the history, and physical examination. Many disappointments have resulted for both patient and doctor from over-dependence on diagnostic skin tests. Not only do *false positive* tests appear with no clinical significance, but also *false negative* tests are common. These latter occur frequently with tests of food antigens, be-

cause the offending antigen is often a digestion product of the food producing symptoms. Thus a negative test with the whole food protein may be misleading. In general, allergy tests are misused. Too many tests are usually done, and some patients are unwittingly misled, because of "all the positive tests". One frequently hears the statement that "so many tests were positive I didn't get any treatment", or the equally discouraging "I had 200 tests but the doctor didn't seem to think they helped". In both cases the patient and doctor gain nothing, and an otherwise useful procedure is discredited. Unless tests are performed properly with active, standardized antigens, and with understanding of the limitation of their usefulness, they are better not done at all. When tests do confirm a clinical impression, skin testing is a useful and valuable guide in the diagnosis of asthma.

TREATMENT:

Treatment will depend on the type of asthma and the severity and duration of symptoms. Avoidance of offending antigens is the most desirable form of therapy, as it is preventive medicine. Unfortunately this is not often possible. Once an attack starts, adrenaline (1:1000) given hypodermically in dosage of 0.1–0.3 c.c. will relieve bronchial obstruction by reducing oedema, and relaxing muscle spasm, and loosening mucus. Other sympathomimetic drugs such as ephedrine, taken orally, will have similar and often more prolonged effect. Combinations of ephedrine with phenobarbital and aminophylline are frequently prescribed. The first two drugs are useful given together, but aminophylline is best given in a solution, rectally, or in a suppository. When an attack has persisted for several hours, the major source of obstruction is mucus plugging the fine terminal bronchioles. Often this mucus is inspissated, and

liquifaction can best be encouraged by the oral administration of saturated aqueous potassium iodide. This is unpleasant tasting, and sometimes causes gastric irritation. It is best given 5–10 drops in water or milk, after meals. It is extremely useful in cases where persisting bronchitis is a problem.

If an attack is not relieved quickly with these standard medications, persisting asthma may lead to the difficult clinical condition described as "status asthmaticus". This gradually increasing degree of asthma usually results from infection involving the bronchi. Patients soon become dehydrated as they are unable to take in their usual requirements of food and fluid. They are working hard at breathing, and also losing vapour in their increased respiratory efforts. Mucus plugging soon becomes the major problem of therapy. Patients will inevitably become restless and anxious, particularly if they have had previous attacks of "status". Sedation is desirable, but morphine is absolutely contraindicated due to its depressing effect on the respiratory centre, and long acting barbiturates often just make the patient feel doped. The most effective sedation is achieved with the rectal administration of ether and olive oil, 2 ounces of each, given rectally, and repeated if necessary. In addition to resting the patient, ether is excreted through the lungs and appears to aid in liquifying bronchial mucus. When the patient is starting to relax, urgent attention should be given to restoring fluid and electrolyte needs. Intravenous infusion of 5% glucose in distilled water can be run in quite freely in the absence of heart failure. Patients may require 3 or 4 litres of fluid in 24 hours to restore proper balance.

Oxygen therapy is not always of real assistance to the patient. They often feel smothered in an oxygen tent. Raising the concentration of oxygen does not seem to greatly relieve the anoxaemia due to

severe bronchial obstruction. If the use of oxygen seems imperative, administration by nasal catheter is better tolerated.

The use of adrenal steroid drugs (cortisone, hydrocortisone, prednisone, prednisolone), and A.C.T.H., have greatly improved the immediate outlook for the patient in status asthmaticus. Where intravenous fluids are required, one flask containing 25 units of A.C.T.H. can be administered slowly over 8–10 hours, while the remaining fluid is given quickly through another set. When the patient starts to improve, and is taking food and fluids readily by mouth, the adrenal steroid drugs can be given orally for a period that will vary in each case. The action of all these compounds is to temporarily suppress inflammation, and hence relieve bronchial obstruction. They in no way replace the need of the patient for standard medication and care. Indeed, infection may not be obvious, clinically, in early status asthmaticus, and adrenal steroid therapy may aggravate the infective process, unless adequate antibiotic or chemotherapeutic drugs are given at the same time. If a patient is sick enough to require hospitalization for treatment of status, and if adrenal steroid drugs are used, it is now the common practice to exhibit antibiotic at the same time. Because of the often dramatic response to such therapy, patients are tempted to believe their asthma problem has been “cured”. This impression should be firmly corrected. One should explain that these most useful agents are available for urgent treatment when needed, but that the proper understanding of all the factors contributing to the development of *status* is paramount. This means a detailed but clear explanation of the various factors present in the problem, and repeated reassurance that the problem can be managed satisfactorily. Most patients are extremely cooperative, and soon learn to help themselves, if such a positive and encouraging attitude is

adopted by the doctor. One of the crucial points is the early recognition of respiratory infection, and early consultation with the family physician for the proper medication. Much of the anxiety and tension associated with recurring asthmatic attacks can be obviated by sympathetic attention by the physician. It is not difficult to appreciate the real fear of attacks that severe asthma patients have particularly when these have occurred before the newer drugs were available.

In cases of asthma that are less severe, and less frequent, *status* may never occur. Frequently such patients have a few inhalant sensitivities, and treatment by avoidance or hyposensitization therapy is most gratifying.

The term hyposensitization is used in referring to the procedure used to give these patients temporary protection by increasing their tolerance to offending antigens. A good example is asthma from contact with grass pollen, occurring in June and July. By administering slowly increasing doses of a standardized solution of grass antigen over 3 to 4 months, the patient's tolerance is raised. The mechanism of this protection is thought to be due to production of univalent (or “blocking”) antibody which can be demonstrated in the patient's serum. Certainly it is not due to “desensitization”, as the titre of the patient's allergic antibody (or “reagin”) is unaltered, or even temporarily elevated, after treatment. The technique of hyposensitization is relatively simple if careful attention is paid to dosage schedules and instructions. There is great individual dosage variation, but once this level has been defined, continuing therapy is straightforward.

It is hoped these few comments will help point up the importance of viewing each patient with asthma as a complex, but not necessarily insoluble problem.

LOUIS — A GREAT PHYSICIAN

W. B. SPAULDING, M.D.

PIERRE-Charles-Alexandre Louis was born in 1787 at Ai, a little town in the province of Champagne, renowned for its wines. He came from a family who had grown grapes and sold wine for generations. Scientists and physicians do not appear in the family tree, though Louis' painstaking methods of clinical observation more closely resemble the watchful habits of expert winegrowers than the careless dogmatism of most XVIIIth century medical leaders. Louis' father died when the boy was only 6 years old. His mother was strict, energetic and intelligent, characteristics which were to become evident in the son. At first his education was supervised by an aunt who had been governess in a French noble family. Later he was tutored by an old monk, a classical scholar. For a brief period Louis studied law but soon he turned from this to medicine. The first medical training was by apprenticeship, visiting in homes and in the hospital with a surgeon who practised in a town nearby and was a friend of his mother.

Like many French medical students Louis settled in Paris for undergraduate training. He must have been a frail lad for three times he was rejected from military service on medical grounds, at a time when Napoleon desperately needed soldiers. Possibly in the interests of his health a friendly doctor dissuaded him from applying for a hospital post. Louis also regretfully gave up the study of an epidemic of pneumonia then raging in Paris. For the doctorate thesis he produced a review of various physiological and pathological subjects, a patch-work of borrowed knowledge of little value because it contained no original observa-

tions. One prophetic gleam flashes out, however, for he wrote ironically, "The only certainty lies in facts. One might say that medicine unlike the other branches of science has its own rules of proof." This jibe protests against the unscientific assertions often made by his teachers and other eminent doctors to defend their personal theories, theories which frequently served not to bind facts together but to distort facts and deny valid observations.

At the age of 26 when he had obtained his medical degree, Louis attempted to make his living by practising in Paris. Unknown and lacking the support of a wealthy family, he failed to establish himself. Louis realized that he must leave Paris and he returned home to consider various plans. Should he go to Constantinople where there were opportunities for young doctors? As fate would have it, precisely at this time of uncertainty, a nobleman named St. Priest visited the family home. The nobleman was returning to Russia to resume his duties as Governor of one of the provinces. He quickly learned of Louis' predicament and had little difficulty in persuading the young doctor to go to Russia with him. The very next day Louis set out for Odessa with what little money his family could scrape up at the last minute. Thus began 6 years so different from his later life that we can only marvel at the contrast between this romantic interlude and the later years of dedicated, selfless clinical work.

During the first years in Russia he travelled widely, summer and winter, making trips which he enjoyed even when a pack of hungry wolves chased the sleigh! With this type of life came

better health, so much so that despite long years of labour, he was nearly 80 years old before he knew what it was to feel tired. Russia proved attractive and exciting. Years later he recalled walking over a frozen lake, plunging suddenly through the ice and almost perishing in the freezing water. After 3 years he settled in Odessa where success in practice soon followed. French émigrés, numerous and influential, sought out the hard-working, conscientious, intelligent young doctor. His clientele and reputation grew until he was appointed one of the physicians to the Emperor. Yet this picture of popularity and beginning affluence was suddenly shattered. Louis encountered a devastating epidemic of diphtheria in which many children lost their lives despite his efforts. After the epidemic had passed he began to wonder whether newer, more effective treatments had been introduced at the great medical centres. Perhaps news of these treatments had failed to reach distant Odessa on the shores of the Black Sea. To dispel the gnawing uncertainty he must find out for himself.

Suddenly the practice is abandoned. Back to Paris and medical studies goes the 33 year old physician. For 6 months he attends the teaching sessions of the children's hospitals seeking the latest knowledge. But what new advances have been made since he graduated? Virtually none. Broussais, at the height of his reputation, has a great following. Students listen while he attributes all pathological processes to "irritation" a word which to Broussais speaks volumes but in fact means nothing. Gastric irritation underlies all diseases, he preaches; tuberculosis, enteritis, these and other conditions stem from a gastritis. Treatment consists primarily of bleeding. The breeding of leeches becomes a major industry with Paris one of the most profitable markets; each year one hundred

thousand leeches suck blood on Broussais wards alone, and at the peak of the craze thirty-three million are imported into France annually. More leeches than citizens! Louis, repelled by the doctrines of Broussais, determines to learn for himself the behaviour of disease. Renouncing practice, he embarks on a period of hospital work unheard of in those days and unusual even by present standards.

To obtain proper perspective we must remember that by the early XIXth century much valuable work had been done by men who were dissatisfied with the superstitions, dogmas and impressions, which passed as medical knowledge. Over a century before, Sydenham had kept careful records of bed-side observations from which he drew memorable pictures of some diseases. For example he gave a graphic description of gout, distinguishing it from a variety of inflammatory conditions within and about joints,—a description so accurate that even to-day it may be read with profit and to the detriment of many modern textbook accounts. In the XVIIIth century Morgagni had founded the science of pathology. Laennec, who followed patients with thoracic disease from the ward to the autopsy room, came to understand so well the findings revealed by his discovery, the stethoscope, that his writings rank as medical classics. Louis set about to apply the methods of such men. He determined to obtain the details of each clinical history with scrupulous care, to keep accurate, unprejudiced records concerning all factors which might bear on the illness, and then personally to perform a complete post-mortem examination on all patients who died. He obtained the permission of his former class-mate M. Chomel to examine all patients admitted to Chomel's service of 46 beds and to perform all the post-mortems. For over 6 years Louis lived in a small room

in La Charité Hospital in Paris. His waking hours were spent questioning and cross-questioning every patient, keeping orderly records and performing autopsy investigations, each one of which took at least 2 hours. In these post-mortem examinations he scrutinized all the viscera in such detail that each inch of gastro-intestinal mucosa was inspected, the larynx invariably examined, the lungs sectioned repeatedly, the central nervous system always searched for lesions. Of course in those days before tissue microscopy all pathological diagnosis depended on the interpretation of changes by the naked eye. One must read the writings of men such as Louis to appreciate their mastery of the subject. Even to-day Louis' book on phthisis stands as an accurate, comprehensive account of the symptoms, physical signs, prognosis and gross tissue changes in advanced tuberculous infection. It's all there—dated 1825—over 50 years before Koch discovered the tubercle bacillus!

For more than 6 years Louis worked. A few understood, like his friend Chomel who urged the reserved, single-minded man to publish his first observations. Most ignored or jeered at the tall, awkward-looking, intense eccentric whom they judged to be wasting his time on public wards and at the autopsy table when he could have been acquiring wealth and prestige. At one point, when Louis had collected observations on many patients with "intestinal fevers" of various sorts, including 50 studied at autopsy, he left the hospital to sort out the distinctive features of these diseases. Paris, too distracting and expensive, was given up for a room in a boarding-house in Brussels. For a year all he saw of Brussels was the neighbourhood restaurant. During the first 4 months he drew up tables of the symptoms, signs and autopsy findings, determining the incidence of each finding, its place in the

natural history of the "intestinal fevers" and always controlling the conclusions by comparison with observations in other types of acute disease. For 8 months he analyzed the tables and put the results in book form. From this came an outstanding account of typhoid fever (the name chosen by Louis) in which he stressed the importance of the lesions of Peyer's patches and demolished Broussais' teachings about the causative role of gastritis. When Louis returned to Paris he was 40 years of age, penniless, but on the way to lasting recognition. For the first time numerical methods had been applied to clinical data and a new research tool, medical statistics, had been introduced. It is for this contribution that Louis is chiefly remembered in the history of medicine.

Little more will be said of his other writings. With Trousseau he thoroughly investigated an epidemic of yellow fever in Gibraltar in 1828. The result—a good clinical and pathological account refuting those who believed the disease was non-infective, showing that one attack usually conferred immunity, and emphasizing that the only consistent lesion was in the liver. Louis destroyed the claim of Broussais that bleeding was the best treatment of pneumonia. He showed that the results of bleeding on Broussais' own wards were not only no better, but were actually worse than the results of treatment without bleeding. The cold figures more than matched the fire of Broussais' outbursts.

Students and assistants have left glimpses of the character and habits of Louis. We learn that he never made a hasty diagnosis: first he would elicit from the patient all the symptoms and signs in masterly fashion; then he would consider all possible diagnoses and all the manifold objections to the diagnostic possibilities; finally, when the fact permitted, he would state his diagnosis. No

snap diagnoses—no opinion based on a single similar case seen years ago! He used to quote Rousseau in words which might have been his own medical creed, "I know the truth lies in the facts, and not in the mind that judges of them; the less I introduce what is merely my own into the deductions I make from the facts the more certain I shall be of approaching the truth." Away with wishful thinking and pet hypotheses!

One student wrote, "To practice percussion well is no easy thing, and no ordinary accomplishment. How often do men, or women, pay 500 dollars to learn to touch the piano; I would willingly pay 500 to anyone who would teach me to percuss like Louis." This promising lad, James Jackson Jr. of Boston, impressed Louis in a way few medical students have ever impressed their teachers. After Jackson had attended the teaching rounds for some time Louis asked, "What is the name of the young American who attends my visits? I mean the one who discovers everything I do when I auscultate our patients." And about the same time he singled out Jackson to come forward to examine the patient. "For" he said, "You are great in auscultation." They soon became firm friends. Jackson, in one of his letters home, writes about a cholera epidemic. He mentions accompanying Louis before breakfast to visit the home of a patient so that they could "learn as exactly as possible the circumstances of the case" from the patient's wife. Jackson describes how the horror of this epidemic affected his teacher, "Never shall I forget Louis' altered face and aspect, like one who had received a blow from which he had not recovered. There are few men living so familiar with death or the dead."

Another American, Henry Bowditch, was stricken with acute rheumatism in his room in Paris. Louis always noted absentees among his students and at-

tended any who were ill. Years afterwards Bowditch recalled, "How like pleasant dreams come up before me the daily morning visits of that excellent man during those weeks of lonely and severe suffering! How genial and even merry his voice at those times! He always had some badinage to cheer me and often words of wisdom." Louis urged Bowditch to make careful notes of his own case pointing out his matchless opportunity to study the disease. Bowditch kept a record until his wrists became so painful that he could not write. Louis then suggested that he dictate the findings! This was too much for even such a keen student as Bowditch.

One clinical assistant never forgot the delicate solicitude with which Louis looked after the coachman of a rich Parisien family. The coachman, admitted to the male ward of L'Hotel Dieu, confided to Louis that in actual fact "he" was a woman and a mother. This woman, some years before, had literally taken over the reins and whip of her dead coachman husband to provide her children with better education. Louis' kindness was not apparent to all; on first acquaintance he seemed haughty and reserved. In the lecture theatre he was sometimes tremulous and uncertain, but all hesitancy vanished at the bedside where his talk fascinated eager students.

Romance and marriage came unexpectedly. A young lady of titled Parisien family consulted the eminent physiologist Magendie about choosing a physician. So many were the outstanding qualities she expected that Magendie told her she wanted a phoenix not a physician. However he recommended Louis as the man who came closest to the requirements. She went to the hospital with her young sister and there met the big, serious man, then in his forties. His friendship with the family ripened, particularly with the younger sister who later became his wife.

After marriage he continued to live without show and without interest in the usual social amusements. The only holiday was an occasional visit to the fishing town of Dieppe. Louis had only one child, a beloved son; the family made up a tight, self-sufficient unit.

One day when the son was 18 and beginning the study of medicine he horrified his father by describing how he had coughed up blood. Who could know better than Louis the ominous significance of haemoptysis in a young person? Indeed his book on phthisis describes fatal cases in which haemoptysis was the first serious portent. In those days tuberculosis was usually diagnosed in a terminal stage and, as Louis had demonstrated in patients under his own care, no effective therapy was known. However, certain places were reputed to have a health-giving location; to one of these in southern France the grieving parents took their boy. Bravely they nursed him, month after month, talking optimistically, whiling away the hours by reading to him but always knowing it was hopeless. Nine months after the first warning he died.

One might imagine that a man of Louis' background and strength of will would have been able to weather this disastrous storm. Not so. Old—now 67, never a man of broad interests, he lapsed into melancholia—sad, always sad—perhaps even enjoying the state, for he copied out the words of Balzac, "It seems to me that sadness takes the place of my friend; I possess it (sadness) with such sweetness, and am so jealous of it, that I believe there would be a second loss if I no longer had it to support me." Each morning he would visit the cemetery to stand patiently beside the grave of his son. He re-read all the books they had shared, marking passages which recalled pleasant discussions. The consulting practice dwindled; no new investigations

were undertaken. Patiently the father waited, strong in the belief that death would bring re-union with his son.

For 17 years more he lived on, less active, but still capable of many charitable acts for Louis believed in charity as a virtue to be practised but never advertised. On one occasion he advanced enough money to enable a contemporary to publish a valuable medical work. The book achieved success, the author prospered, but the debt remained unsettled. When the author died Louis tore up the promissory note to avoid embarrassing the dead man's relatives. Another time he left an unusually generous endowment to an association organized to aid doctors who had become unable to support themselves.

No better words could conclude than those which Louis read out when Bowditch was about to leave Paris. "There is something rarer than the spirit of discernment, it is the need of truth; that state of the soul which does not allow us to stop in any scientific labours at what is only probable, but compels us to continue our researches until we have arrived at the evidence." Louis as much as any man was possessed by that rare "state of the soul".

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CHEST PAIN, A PROBLEM IN DIAGNOSIS

M. A. TYBER 5T8

Mr. J. S., age 43, admitted to hospital July 13, 1956.

CHIEF COMPLAINT: Left lower chest pain of 1½ months' duration.

HISTORY OF PRESENT ILLNESS: Two years ago, and again one year ago, he experienced episodes of fever, nausea, and vomiting each lasting about 14 days. A G.I. series at the time was negative. Two months ago, he noticed malaise, fatigability and feverishness with a mild cough productive of small amounts of white sputum. Three weeks ago, the cough became more severe, and he was forced to stop working as an attendant at a gas station because of knife-like pain in his left chest and shoulder which was aggravated by deep respiration or coughing. At this time, he developed anorexia and vague generalized pains.

A few days later, the pain abated, and he returned to work. Because of the continuing malaise, he consulted a physician who advised hospitalization.

At no time did he have hemoptysis or purulent sputum. There was no history of previous pleurisy or pneumonia, or contact with T.B. In the past three weeks before admission, he lost 18 pounds.

PAST HEALTH: Measles and mumps as a child. Occasional epigastric burning of 2 years' duration, occurring once or twice monthly at no specific time. Gall bladder

investigations done a year ago were negative.

PERSONAL HISTORY: Married for 10 years but has no children. No familial history of diabetes or T.B. Drank 4 pints of beer daily but stopped 3 years ago due to G.I. symptoms. Smokes 2 packages of cigarettes daily.

FUNCTIONAL INQUIRY:

General: Tired and weak. Recent weight loss. No night sweats or chills. No allergies.

Head and Neck: good vision.

Ear, Nose and Throat: no complaints.

Neck: no pain, swellings, or stiffness.

Respiratory: As in H.P.I.

Cardiovascular: No decreased exercise tolerance, no dyspnoea, no swelling of ankles, no history of rheumatic fever.

Gastro-intestinal: anorexia past 4 weeks. No dysphagia, indigestion, hematemesis, melena or jaundice. No herniae or hemorrhoids. No change in bowel movements.

Locomotor: No complaints.

Central Nervous System: No headaches, dizziness, fits, paralysis or paresthesiae.

PHYSICAL EXAMINATION:

General: 43 year old white male with pallor and evidence of recent weight loss.

Head and Neck

Eyes: pupils equal and reactive to light and accommodation. Fundi clear.

E.N.T.: clear.

Neck: no palpable glands.

Respiratory: Decreased movement of left hemithorax. No clubbing of fingers. Trachea mid-line. Decreased vocal fremitus of left chest posteriorly and laterally. Dullness to percussion and decreased breath sounds over the same area. No rales or rhonchi.

Cardio Vascular: B.P. 100/60. Pulse 96/ min., regular. Heart not clinically enlarged. No murmurs.

Abdomen: Abdomen scaphoid with some guarding. Liver and spleen not palpable. On rectal, there was good sphincter tone, with no masses or tenderness.

Central Nervous System: Cranial nerves intact. Joints and muscles normal. Sensation good for light touch, pin-prick, and position sense. Reflexes physiologic.

Differential Diagnosis:

Left-sided pleural effusion due to

- I) post-pneumonic state
- II) T.B.
- III) malignancy
- IV) pulmonary infarction.

PROGRESS IN HOSPITAL:

July 13: 400 c.c. amber fluid aspirated from left chest. sp. gr. 1.022; Gram and Ziehl-Neelsen negative. Cytology of aspirate showed lymphocytes, polys, and histiocytes.

July 14: Chest films after aspiration showed a high diaphragm on the left with a compression atelectasis of the left lower lobe. The hilar regions were normal.

July 16: For the first time a bulge was noticed anteriorly in the left upper quadrant. A mass could be palpated here which did not ballott and was

associated with increased dullness in Traube's Space.

July 18: Flat plates of the abdomen revealed a homogenous soft tissue mass without calcification depressing the splenic flexure and left kidney. The mass was thought to be an enlarged spleen.

July 23: Barium enema revealed no colonic lesion and an I.V.P. showed the mass was not a kidney.

Aug. 2: A surgical consultation was arranged and the following differential diagnosis was proposed:

- pseudo pancreatic cyst
- subphrenic abscess.
- retro peritoneal tumour.
- ruptured aneurysm of Splenic artery.

The patient was transferred to a surgical ward and an aortogram showed the splenic artery passing to the mass. There was no concentration of the dye or vascularization in the mass.

Aug. 13: An upright film of the abdomen was done which again visualized a mass and showed a fluid level in it. It was therefore thought the mass was spleen associated with a cyst.

LAB WORK:

With the exception of an elevated serum amylase, routine laboratory examinations were normal.

Aug. 21—An exploratory operation was performed via an abdomino-thoracic approach. A fairly large cyst was found, partly adherent to the inferior surface of the diaphragm, as well as to stomach and small bowel. Dissection was carried out down to the anterior surface of the tail of the pancreas. The cyst was freely separated without exposing raw viable pancreatic tissue. The wound was closed with intercostal drainage and underwater seal and a rubber drain in the left upper quadrant.

The post-operative course was uneventful with eventual full re-expansion of the left lung and no fistula formation.

SURGICAL-PATHOLOGICAL REPORT:

The cyst measured 17 cm. in diameter and was full of dark brown fluid. An interesting finding was that the spleen was incorporated in the cyst wall. Microscopic examination showed areas of fat necrosis and old and recent hemorrhage. No pancreatic tissue could be identified.

On the basis of the high serum amylase, the fat necrosis, and the hemor-

rhage, the pathological diagnosis was "pancreatic cyst".

SUMMARY:

This patient presented with a left pleural effusion and a two year history of epigastric distress. A mass was discovered in the left upper quadrant and after removal, the patient made a complete uncomplicated recovery.

BRONCHIECTASIS

N. P. HUSCHILT, 5T7

DEFINITION:

Bronchiectasis means dilatation of the bronchi. It refers to a bronchopulmonary disorder characterised by a permanent pathological dilatation of the bronchi, within which, infection is a frequent complicating factor.

INCIDENCE:

Bronchiectasis is a fairly common disease. At present it may be next to tuberculosis as a cause of chronic lung disease. The autopsy incidence has been stated to be between 0.4 and 4%, and the clinical incidence, about 1%. Wynn-Williams found unequivocal bronchiectasis, proved by bronchogram, in 166 patients from a clinic which was responsible for 150,000 people in the Bedford area of England. Other cases of definite bronchiectasis were discovered, though not confirmed by bronchogram. Wynn-Williams counted a total of 200 cases living in the area at the end of 1951—an increase of 1.3 per 1000. No case of bronchiectasis arising as a complication of more serious disease was included—and hence bronchiectasis associated with conditions such as carcinoma of the bronchus or post-primary tuberculosis was not considered.

Perry and King have demonstrated that bronchiectasis is principally a disease which starts in childhood and early adulthood. The age of onset in 42% of their cases was under 10 years of age, 27% were between the ages of 11 and 20 years. More than two-thirds of their cases therefore, had their first difficulty prior to the age of 20 years.

ETIOLOGY AND PATHOGENESIS:

Various factors play a part in causing bronchiectasis. These may be divided into two main groups; congenital and acquired.

Congenital: The congenital causes are relatively insignificant. Congenital cystic disease may be complicated by infection and present as bronchiectasis. Kartagener's Syndrome or triad, which includes situs inversus, sinusitis and bronchiectasis, is due to a congenital maldevelopment. Fibrocystic disease, or mucoviscidosis, predisposes to chronic lung infection, including bronchiectasis. Intralobar pulmonary sequestration presents itself with symptoms and signs similar to those of bronchiectasis.

Acquired: In Wynn-Williams' series, pneumonia was judged to be responsible for 43%, whooping cough and the in-

fectious fevers for 16%, recurrent bronchitis and cough since infancy for 21% and primary tuberculosis for 7%. A number of miscellaneous causes make up the totals. Mallory, after reviewing several series, reports that several histories of pneumonia or of upper respiratory infections complicated by pneumonia, such as measles, pertussis and influenza are obtained in 50% or more of the patients.

The pathogenesis of bronchiectasis is often complex. Boyd cites two factors; bronchial obstruction and infection. Sputum may seal off a major bronchus and produce atelectasis in that part of the lung supplied by the bronchus. This plug tends to move peripherally so that ultimately most of the finer bronchi become sealed, while the proximal part of the bronchial tree again becomes patent. *Two mechanical forces now operate tending to produce bronchial dilatation.* The pressure of the atmosphere within these open bronchi and the pull of the lowered intrapleural pressure transmitted through the atelectatic, solid lung, supply the forces needed for dilatation.

Permanent bronchial dilatation will not occur in the absence of destructive changes in the bronchial wall. These changes come about as a result of chronic inflammation. In the dilated bronchi, pooling of bronchial secretions occurs. This serves as an excellent culture medium for bacteria. The infected bronchial material causes inflammation of the bronchial wall. As the condition persists, the elastic fibres and musculature in the bronchial wall are replaced by inelastic fibrous tissue. Provided the atelectasis continues, the forces tending to produce dilatation will persist, and eventually this dilatation becomes permanent. This type of bronchiectasis tends to be segmental or lobar. Other causes of bronchial obstruction are enlarged hilar lymph glands, intrabronchial tumours, aspirated pus and foreign bodies. These

may all cause bronchiectasis in the same way.

Another type of bronchiectasis appears to begin with an acute infection such as measles, whooping cough or influenza. The distribution of bronchiectasis in this instance, is patchy rather than segmented. Destruction of the bronchial wall by the initiating infection is thought to render it distensible by the ordinary forces of respiration.

PATHOLOGY:

Gross: The most striking feature is the dilatation of the bronchi. These dilations may be cylindrical or saccular, the former being more common. The bronchi may be filled with pus and the peribronchial area may show a localized consolidation. The mucosa is swollen, edematous and redundant.

The pathological changes are not confined to the bronchial walls. The surrounding lung parenchyma involves the peripheral bronchi. One segment only, or many segments of the lung may be involved in one or more lobes. It is more often unilateral than bilateral, and involves the left side more than the right. Obstructive atelectasis is most likely to develop in the lower or middle lobes and in the lingula of the left upper lobe—the same distribution in which bronchiectasis is most common.

Microscopic: The microscopic appearance is a combination of inflammation and degradation of tissue. It will vary with the cause and chronicity of the process. The bronchial epithelium shows various degrees of inflammatory change. Ulceration, squamous metaplasia and destruction of cilia are seen. The submucosa is edematous, congested and shows a considerable degree of round cell infiltration. There may be septic thrombosis of the veins.

The bronchial walls in advanced cases show destruction of the elastic fibrillae,

smooth muscle fibres and cartilage and replacement by fibrous connective tissue. The peri-bronchial parenchyma may show atelectasis, pneumonitis, chronic inflammation or frank suppuration.

The pathological picture in bronchiectasis accounts for the disturbance in the normal clearing mechanism of the bronchi. This is due to the destruction of the elastic elements in the bronchial wall and the loss of cilia due to squamous metaplasia, ulceration and destruction of the bronchial mucosa. As a result, the important functions of bronchial contraction and ciliary activity are lost, and the patient's ability to deal with infected bronchial secretions is impaired. An obliterating endarteritis of the vessels in the bronchial wall may still further lower the resistance. This tends to make bronchiectasis a chronic, self perpetuating disease.

CLINICAL PICTURE:

The clinical picture of bronchiectasis is very variable. Some of the prominent features will be mentioned.

SYMPTOMS:

Previous infection: Characteristically, these patients give a history of childhood illnesses especially measles, whooping cough, influenza, sinusitis and other respiratory tract infections. They tell of frequent bouts of chest illness which are more common in the winter months. Recurrent attacks of pneumonia are common.

Cough: Chronic cough is a common complaint. This cough is typically worse in the morning and is influenced by changes in posture. It may be paroxysmal and there may be no relief until sputum is coughed up and expectorated.

Sputum: The amount of sputum varies greatly but it may be considerable. As much as 100 ml. of sputum is not uncommon. While the patient is asleep, pus

tends to pool in the lumina of the dilated bronchi. With changes in posture, these bronchial contents come in contact with the more proximal and sensitive portions of the bronchial tree. This initiates the cough reflex, and the quantity of pooled bronchial material is coughed up. The sputum may be fetid but usually it is not. Fetid sputum is more common in older patients.

Hemoptysis: Hemoptysis is common but again is variable in amount. Hemoptysis was a prominent complaint in a case of bronchiectasis which Laennec first described in 1819. That it is common in this condition is readily understandable. The bronchial submucosa is congested and the overlying mucosa frequently ulcerated.

Liebow and his workers moreover, have demonstrated that a remarkable enlargement of the bronchial arteries occurs and that a rich anastomosis with the pulmonary arteries develops. This tends to produce pulmonary hypertension and ultimately to the development of cor pulmonale.

Hemoptysis in bronchiectasis varies from streaking to gross hemorrhage. Some cases of total hemorrhage have been reported.

Dyspnoea: This is seen in the severe cases. 20% of the cases of bronchiectasis in Wynn-Williams' series had dyspnoea on slight exertion.

Constitutional Symptoms: Pneumonitis and suppuration may complicate bronchiectasis and cause chronic toxemia. The patient complains of a general decline in health associated with lassitude, anorexia, night sweats, weight loss and fever. In some cases, bronchiectasis may present as one of its complications: heart failure, brain abscess, empyema or amyloidosis.

SIGNS:

There are no physical signs either in the chest or elsewhere which are pa-

thognomonic of bronchiectasis. Abnormal physical signs in the chest are present in a high proportion of cases, the most typical being persistent moist rales localized to the damaged part of the lung. These abnormal signs are usually confined to the lower lobes and are therefore, best found posteriorly. They depend on the amount of secretion in the bronchiectatic spaces, the air entry and on the nature of the change in the parenchyma near the affected bronchi.

Atelectasis: There is diminished movement in the involved part of the lung. The trachea may be shifted toward the affected side. Breath sounds are often diminished.

Partial obstruction of a bronchus: In this case, a rhonchus is heard over the involved bronchus and cough will cause it to alter or disappear.

Secretions in the bronchiectatic spaces: Rales are heard which are most commonly present over the lung bases posteriorly. They may be post-tussive and heard only after a prolonged, forceful expiration followed by a cough.

Consolidation: There is dullness on percussion and the breath sounds are bronchial.

Clubbing of the fingers and toes: This may develop in conjunction with hypertrophic pulmonary osteoarthropathy. It is uncommon but may be seen in severe cases.

SPECIAL INVESTIGATIONS OF BRONCHIECTASIS:

Plain X-ray film of the chest: This is often normal especially in uncomplicated cases of bronchiectasis. When bronchial obstruction occurs, there will be atelectasis distal to the obstruction. In the presence of pneumonitis, consolidation is seen. In some cases, there is an increased prominence of the linear markings of the lung especially of those going to the lower lobes. When filled with air, the

bronchiectatic cavities may be visualized. If these spaces are partially filled with secretion, fluid levels may be seen.

Fluoroscopy: This technique will show deviation of the mediastinum in the presence of atelectasis. The value of fluoroscopy in the diagnosis of bronchiectasis is small.

Bronchography: The development and perfection of this technique has aided greatly in the diagnosis and treatment of this disease. A contrast medium is required to outline the bronchial tree. This is accomplished by the introduction of a radio-opaque liquid into the various bronchi.

The chief changes will be noted in the tertiary bronchi and beyond them. A bronchus may be considered to be abnormally dilated when it is wider than the one proximal to it. Various sizes of bronchi may be seen. The bronchiectatic part may have what has been described as a "leafless tree" appearance. There are thick branches but there is typically an absence of the normal pattern of fine peripheral arborization.

Bronchoscopy: This affords a direct visualization of the bronchial tree. It is used to distinguish the type of bronchiectasis caused by persistent bronchial obstruction from that in which no obstruction exists. It may also show from which lobe or segment most of the pus originates. The object causing the bronchiectasis may be seen, as for example, a bronchial neoplasm or a foreign body.

Sputum examination: Sputum smears and culture are used to determine the type of pathogen present and to rule out the possibility of pulmonary tuberculosis. The daily output of sputum should be measured and its character noted. In a typical case, it is thick, purulent and of a yellowish or greenish colour. On standing, it separates into three layers, an upper frothy layer, a middle cloudy layer, and a lower purulent layer.

DIFFERENTIAL DIAGNOSIS OF BRONCHIECTASIS

Chronic bronchitis or infective asthma: The physical signs in the chest tend to be symmetrical and bilateral. Foul smelling sputum and clubbing of the fingers are not seen. The bronchogram shows no bronchial dilatation.

Pulmonary tuberculosis: The affected portion of the lung is usually apical rather than basal. The sputum may show acid fast bacilli.

Suppurative Pneumonia: The onset is usually abrupt. Sputum may be slight or absent. There is no history of a chronic cough which is influenced by postural changes. Pneumonia may precede bronchiectasis, however, or may be a complication of it.

Chronic draining lung abscess: In this condition, the patient has symptoms similar to those in bronchiectasis. A plain X-ray of the chest may show a cavity with a fluid level.

Carcinoma of the Lung: Weight loss, anemia and an unproductive, hacking cough may be present. Recurrent pneumonia and chest complications may obscure the diagnosis. Bronchoscopy and a plain X-ray film of the chest may demonstrate the tumour.

Chronic Sinusitis: A chronic cough with purulent sputum and repeated "colds" caused by chronic sinusitis may be difficult to differentiate from similar symptoms in bronchiectasis. Physical signs in the chest will be absent in uncomplicated chronic sinusitis and the bronchogram will not show dilated bronchi. Sinusitis and bronchiectasis, however, may co-exist.

COMPLICATIONS OF BRONCHIECTASIS:

Recurrent Pulmonary Infection: The pooling and stagnation of bronchial secretions predisposes to chronic infection within the bronchi. With extension

of this infection to the lung parenchyma, the patient develops pneumonia or an acute "flare-up" of his condition.

Congestive Heart Failure: An extra load is thrown upon the heart as a result of the anastomosis between the bronchial and pulmonary arteries, and the emphysematous change which the lung parenchyma may undergo.

Empyema: This will occur when supuration extends to and bursts into the pleural space.

Metastatic Cerebral Abscess: This is a grave and fortunately rare complication of Bronchiectasis.

Sinusitis: Bronchiectasis may co-exist with chronic sinusitis. The latter may clear when the bronchiectasis is treated.

Amyloidosis: Amyloidosis may follow any chronic suppurative condition. It rarely occurs with bronchiectasis.

Respiratory Failure and Emphysema: Where intrabronchial obstruction is incomplete and expiration is impeded more than inspiration, the alveoli tend to dilate, and emphysema results.

Hemoptysis: This has already been mentioned as a frequent complaint. Occasionally it is very serious.

TREATMENT

The treatment of bronchiectasis may be medical or surgical. The choice of treatment will depend on the degree of the patient's disability, the extent of lung involvement, his pulmonary reserve, the presence of other associated disease, particularly infective asthma and sinusitis, and the general state of his health.

Medical Treatment. This form of treatment will not reverse the bronchiectatic changes. This is its major limitation. Medical treatment is intended to be palliative and is directed toward minimizing infection. The objectives of medical treatment are:

- 1) adequate endobronchial drainage

- 2) eradication of bacterial invaders by chemotherapy
- and
- 3) improvement of the patient's general health.

Clearing of the bronchial tree is accomplished by postural drainage, and the use of expectorants. The patient is told how to place himself so that bronchi draining bronchiectatic segments are as vertical as possible. Good drainage may be achieved by coughing while in this position. This procedure is repeated at regular intervals. Sticky, bronchial secretions may be made less tenacious by the use of expectorants such as potassium iodide. Steam and hot drinks help the patient to cough up sputum.

Antibacterial agents are essential in the management of acute exacerbations. The organisms most frequently found in bronchiectatic sputum are pneumococci, hemolytic streptococci, staphylococci and hemophilus influenza. Spirochetes and fusiform bacilli are sometimes seen. Penicillin, given orally or intramuscularly is probably the most effective antibiotic. The bacterial flora is often mixed. In this case, a broad spectrum antibiotic may be used.

Definite measures are indicated to improve the patient's general health. Other foci of infection, such as tonsils, sinuses and adenoids should be treated. Adequate rest and nutrition are essential. A warm, dry climate often helps to alleviate a patient's symptoms. Occasionally, this change alone will cause the patient to be free of sputum.

Surgical Treatment. Surgical excision of the bronchiectatic portions of the lung is the treatment of choice when the operation can be performed with reasonable safety and without too much embarrassment of pulmonary function. The determination of the exact extent of the disease by bronchography is essential

before operation. Surgery is reserved for patients whose disease is so localized that excision of the involved part can be accomplished without causing respiratory failure. The maximum amount of tissue that can be removed is one entire lung or portions of two lungs which together do not equal more than one entire lung.

The mortality and morbidity associated with surgery has decreased markedly with the introduction of antibacterial agents and the improvement of surgical technique. The development of hilar dissection and individual ligation of hilar structures, has reduced the post-operative complications, especially broncho-pleural fistula and post-operative empyema. The mortality, in selected cases, is now less than 1%. The operation does not result in thoracic deformity, and little pulmonary disability results if the disease is reasonably well localized. In suitable cases, surgery permanently cures the patient. The operation is best tolerated by children and young adults.

Prophylaxis is the best treatment of bronchiectasis. This consists in early diagnosis and prompt treatment of pneumonia, whooping cough and other respiratory tract infections especially in young people. Foreign bodies in the bronchial tree should be removed as early as possible. Prompt and adequate treatment is essential to prevent the development of permanent bronchiectasis. The most hopeful prophylactic measure in the prevention of bronchiectasis is the prevention or prompt alleviation of atelectasis.

PROGNOSIS

Bronchiectasis in former years carried with it a very gloomy outlook. This picture has brightened considerably in recent years. Antibiotics help to control

the acute exacerbations of the disease and decrease the incidence of complications. Surgery offers the hope of permanent cure to many patients.

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HEMOPTYSIS

J. R. HAMILTON, 5T8

HEMOPTYSIS is the spitting of blood that has originated in the respiratory tract. The term may be applied to the streaking of sputum or to the production of frank blood of any amount. Hemoptysis usually refers to those conditions in which the blood has come from structures below the nasopharynx.

It is important to differentiate hemoptysis from hematemesis. Usually the differentiation presents little difficulty, as the patient will know whether he has coughed, spit or vomited. A confusion of terms may be encountered in cases where there is a language difficulty.

The blood of hemoptysis may be red or altered, free or mixed with sputum. Often, it appears in the patient's mouth without a cough. Blood is relatively non-irritating to the bronchi and may reach the mouth by means of bronchial movements and ciliary action. The patient will complain of a peculiar taste in his mouth. The local wheeze or gurgling, often attributed to hemoptysis, will only be experienced by the patient if the bleeding is profuse.

Hematemesis characteristically produces blood altered by gastric juice and

is said to resemble coffee grounds. However, esophageal hemorrhage or massive gastric bleeding may produce red unaltered blood.

Once it has been established that hemoptysis has occurred, bleeding from the nasopharynx must be differentiated from lower respiratory tract bleeding. Only those conditions causing bleeding from the lower respiratory tract will be considered in this article.

ETIOLOGY

Most diseases or trauma, involving structures in the chest can, conceivably, cause hemoptysis. Only the commoner causes have been included in the following classification.

1. LESIONS OF THE LARYNX, TRACHEA AND BRONCHI
 - A. Inflammation
 - i) tracheobronchitis
 - ii) ulceration
 - B. Neoplasm
 - i) adenoma
 - ii) carcinoma
 - C. Dilated Mucosal Veins
 - D. Trauma

2. LESIONS OF LUNG PARENCHYMA

- A. Inflammation
 - i) tuberculosis
 - ii) pneumonia
 - iii) bronchiectasis
 - iv) lung abscess
- B. Neoplasm
 - primary carcinoma
- C. Trauma
 - i) hemorrhage
 - ii) penetrating wound

3. VASCULAR LESIONS

- A. Pulmonary Congestion
 - i) mitral stenosis
 - ii) left heart failure
- B. Pulmonary Infarction

INVESTIGATION OF THE PATIENT WITH HEMOPTYSIS

History

The patient is seldom examined during an attack of hemoptysis. Frequently the doctor must rely on the patient's own description of his hemoptysis. Certain features of the bleeding are of particular interest.

a) *The frequency* of the hemoptysis is important. Frequent bleeding suggests ulceration or bronchogenic carcinoma while single attacks are sometimes a feature of tuberculosis.

b) *The duration* of bleeding should be determined. Benign, chronic conditions, such as mitral stenosis or bronchiectasis may give a history of hemoptysis extending over several years. The bleeding due to carcinoma will not have a long history.

c) *The colour* of the expectorated blood is important. Red blood suggests a non-infective or tuberculous process. Altered blood is a product of infections that break down hemoglobin.

d) *The quantity* of blood is extremely variable and not of great diagnostic significance. Usually the blood is small or moderate in amount but occasionally any

one of the conditions causing hemoptysis may give rise to massive bleeding.

e) *Symptoms rarely localize a bleeding point in hemoptysis.* Occasionally, when there is a considerable amount of hemorrhage the patient feels a wheeze in the area of bleeding. Bronchogenic carcinoma or lung abscess are capable of producing pain or discomfort in an area of the chest, unrelated to the site of hemorrhage.

Physical Examination

The nose, throat and mouth must be examined thoroughly for possible sources of hemorrhage. There may be no positive findings on examination of the chest. If the patient is examined during, or shortly after the bleeding, rhonchi, due to blood in a bronchus may be heard. In massive hemorrhage, blood may be aspirated into areas of normal lung. Consequently caution must be exercised in placing the lesion in an area where localized coarse rhonchi are heard.

Chest X-rays, both postero-anterior and lateral views, should be taken as soon as possible. The sputum must be examined prior to the institution of antibiotic therapy, determining the presence of any pathogenic organisms or malignant cells, if indicated.

It is often essential to employ bronchoscopy to determine the source of bleeding. The bronchoscope will often produce material for bacteriological or pathological examination.

It may be necessary to use bronchograms or laminography to demonstrate processes such as bronchiectasis, lung abscess or neoplasm.

SOME CLINICAL FEATURES OF HEMOPTYSIS

Bronchiectasis

Ulceration of the bronchial mucosa may cause bleeding in bronchiectasis. Hemoptysis is, at times the presenting complaint. It may be of five or ten years

duration and tends to occur in single widely spaced attacks of two to four days duration. The hemorrhage is usually small in amount but it may be massive. The blood may be mixed with sputum, but it also occurs in the absence of sputum in so-called "dry", non-infected bronchiectasis.

Pulmonary Tuberculosis

Today, tuberculosis is being recognized at an earlier phase than in the past. Modern methods of treatment and early recognition of the disease have resulted in fewer cases of advanced tuberculosis. The cavity of advanced pulmonary tuberculosis seen more frequently in the past, often led to massive hemorrhage because of tuberculous erosion of the larger vessels in the cavity.

In the less severe tuberculosis seen today, bronchial ulceration and necrosis of small pulmonary vessels cause bleeding that is moderate in amount and infrequent. The blood of tuberculous hemoptysis is usually unaltered.

Bronchiectasis may be associated with a healed tuberculous lesion.

Pulmonary Congestion

It is probable that high pulmonary venous pressure causes a dilatation of the distensible pulmonary capillary bed. Extravasation of red blood cells occurs as a result of the associated increase in capillary permeability. The anastomoses with the bronchial vessels are also distended. The swollen, thin-walled vessels are liable to rupture.

The hemoptysis of pulmonary congestion is related to the peaks of pulmonary venous pressure. Therefore, it occurs on exertion. In mitral stenosis it is usually relieved by rest but it may recur after several weeks, months or years. Hemoptysis will be prolonged in cases of prolonged heart failure.

It is not wise to bronchoscope a patient with pulmonary congestion.

Pneumonia

Bacterial pneumonia is frequently characterized by the presence of blood in the sputum. Hemorrhage is not a feature of any of the viral pneumonias. In pneumococcal pneumonia the blood is usually altered to a rusty colour but it is occasionally red. Staphylococcal pneumonia rarely causes hemoptysis. If bleeding does occur the blood may be red or rusty. The sputum of Friedlander's Pneumonia is typically brown. In bacterial pneumonia, the blood is always small in amount and mixed with sputum.

Unusual or prolonged hemoptysis in patients diagnosed as having acute pneumonia is of considerable significance since the symptoms of pneumonia may be the presenting symptoms of other lesions in the lung. Prolonged or recurrent hemoptysis beyond three days in a case of lobar pneumonia, should be carefully re-examined.

Neoplasm

Bronchogenic adenomas tend to have prominent surface blood vessels that may cause profuse hemorrhage.

The hemoptysis of Bronchogenic carcinoma is due to necrosis of the tumour itself or to invasion of blood vessels by the malignancy. Hemorrhage usually occurs every day or two and it is small in amount. Lesions in the larger bronchi show hemoptysis at an earlier stage of the disease than more peripheral lesions. Hemoptysis is usually preceded by a change in the cough pattern, fatigue, and bizarre symptoms in the chest.

Hemoptysis in the Absence of Serious Disease

Often no disease process can be discovered following the thorough investigation of the patient with hemoptysis. A large percentage of these patients when followed over the years, fail to develop demonstrable disease. Various causes have been suggested such as small in-

farctions, blood dyscrasias, small areas of bronchiectasis, sclerosis of small blood vessels or ulcerative lesions in segmental bronchi and their branches. It is possible that hard coughing, per se, may cause bleeding and hemoptysis just as does the insertion of a bronchoscope. Structural or functional defects, causing an increase in capillary permeability, and yet compatible with a healthy life have been suggested as possible causes of benign hemoptysis.

The Significance of Hemoptysis

One cannot point to the commonest single cause of hemoptysis since each paper on the subject differs in the age, geographical distribution and other qualifications of its particular population sample.

In a study made on 1316 patients attended by a thoracic surgeon, Abbot has given us some idea of the frequency with which hemoptysis occurs in some of the commoner diseases of the chest. This group would differ in its makeup from a group consulting an internist. The author has excluded uncomplicated cases of lobar pneumonia from his calculations. Thirty-eight per cent of the group of 1316 patients with chest disease suffered hemoptysis. In 16 per cent of this group showing hemorrhage, hemoptysis preceded any other sign or symptom. Abbot estimates that 85 to 90 per cent of all patients with hemoptysis have a serious underlying disease.

In a group of 302 tuberculous patients, 37 per cent suffered some degree of hemoptysis. 54 percent of 187 patients

with bronchogenic carcinoma, 44 per cent of 239 patients with bronchiectasis and 49 percent of 65 cases of lung abscess all suffered some hemoptysis.

Although approximately 10 to 15 per cent of patients with hemoptysis may be perfectly healthy individuals, it is obvious that hemoptysis is a sign found frequently in many serious diseases of the chest. It is the responsibility of the doctor, when he investigates a patient with hemoptysis to determine, if possible, the origin and the cause of bleeding.

ACKNOWLEDGEMENT.

I am grateful to Dr. W. A. Oille for his interest and assistance throughout the preparation of this article.

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ABSTRACTS

Indications for Treatment of Primary Tuberculosis in Children: E. M. Lincoln, Bull. N. Y. Acad. Med., 7: 509, 1956.

The importance of the general management of the tuberculous child has not been decreased by the recent use of specific drug therapy. Active tuberculosis without symptoms does not necessitate hospital or sanatorium care, nor even any greater general consideration than the reduction of normal physical activity and a liberal diet. Active tuberculosis, however, requires special considerations, regular examinations, and prolonged conservative treatment.

Cases for specific therapy are selected on the basis of their probable prognosis, with meningitis and miliary tuberculosis invariably demanding anti-microbial therapy. Locally progressive primary pulmonary tuberculosis, and protracted haematogenous tuberculosis other than miliary, also indicate anti-microbial therapy. The principal drugs in use are Streptomycin (1.0 Gm. daily I-M), Isoniazid (10 mgm./day orally), Promizole (0.25 — 8.0 Gm./day orally) and P.A.S. (0.5 Gm./kg./day orally, to a maximum 12 Gm. daily dose), with the dosages modified in younger children and infants.

Meningitis and miliary Tuberculosis are treated intensively with Streptomycin and Isoniazid. Protracted haematogenous tuberculosis and locally progressive primary tuberculosis are treated with Isoniazid, often combined with Promizole or P.A.S., with Streptomycin held back in reserve. Treatment is carried on for at least a year, and for three to six months after control of the disease.

Specific treatment, as well as being a life-saving measure, is used to alleviate

symptoms, shorten the duration of illness, and minimize sequelae. Certain complications, however, show no apparent response to specific therapy, and in other complications the value of therapy is difficult to evaluate due to the normal chronic course with spontaneous remissions.

Treatment of asymptomatic primary tuberculosis is solely to prevent complications, but considerable research must be done on the incidence of serious complications in untreated and treated cases before the value of such a large program of therapy can be established. At present, prevention should be of greater concern, where tuberculosis has not already gained entry.

D. C. PRICE

Pulmonary Infarcts and Pulmonary Carcinoma. Balo, J., Juhasz, E. and Temes, J. Cancer. 9:918, 1956.

Most of the current work in the field of lung cancer has involved the investigation of the effects of inhaled carcinogens and dusts. The authors list several investigators who believe they have found a relation between lung scarring and carcinoma.

On the border of some old haemorrhagic infarcts the alveolar epithelium proliferates. The nuclei in these cells tend to be of equal size, but foci of irregular large dark nuclei are seen. Some of the alveolar cells undergo metaplasia and clumps of squamous cells are found. The authors believe that these islands may undergo a frank malignant transformation. Alveolar cell carcinomas may be multicentric in origin. In the material studied, all cases had proven histories of pulmonary infarction. All bodies were carefully autopsied to rule

out the presence of metastasizing carcinoma.

Endogenous rather than the more commonly suspected exogenous factors seem to have played a part in the pathogenesis. The authors suggest that anoxemia, regeneration, and the effect of irritative substances, liberated at the site of necrosis—'necrohormones'—are responsible.

J. A. LOWDEN

The Effect of Corticotropin (ACTH) and Cortisone in Fifteen Cases of Pulmonary Tuberculosis. E. J. Des Autels, J. R. Zvetina, G. S. Berg, J. Fershing, S. Freeman. *Diseases of the Chest*, vol. 30, #5.

Despite the numerous studies that have taken place over the past decade controversy still continues over the use of ACTH and cortisone in the treatment of pulmonary tuberculosis. In assessing the value of these drugs one must remember that ACTH stimulates all of the known functions of the adrenal cortex, increasing the production of catabolic glucocorticoids and that of the anabolic androgenic and oestrogenic hormones, with the single exception of the mineralo-corticoid, aldosterone. Cortisone, on the other hand, suppresses these functions of adrenal cortex and also suppresses the pituitary, thus reducing the production of some of the trophic hormones. The consequent decrease in target gland hormones will interfere more seriously with factors of host resistance than that seen with ACTH.

The response to therapy as seen in this group of patients is divided into two main headings;

(a) Clinical. All of the patients showed a dramatic improvement in the clinical picture featured by an abrupt defervescence of fever and toxæmia, a marked increase in appetite, a gain in weight and an improved sense of well being. Sudden

discontinuation of therapy for any reason resulted in an immediate recurrence of the former symptoms, frequently in a more severe form.

(b) Roentgenographic response. Serial X-rays demonstrated progression of the disease in 12 cases, no change in 1 case, and slight improvement in 2 cases. Three patients died during therapy. Progression of the disease was often marked and showed a tendency to extensive cavitation and to spread to new areas of the lung.

(c) Survival. The end result was that 10 died and 5 survived—of these three are alive and well, two are still hospitalized four years after hormone therapy.

Summary. Cortisone was always a harmful drug, and although ACTH may be indicated in the occasional case, it is usually contraindicated. The indications for hormone therapy in association with pulmonary tuberculosis are always relative rather than absolute, and in special cases the advantages may outweigh the disadvantages. In any event, rigorous antimicrobial therapy must be employed if these steroids are to be used.

R. IAN HECTOR
IV MED.

Cortisone and Chemotherapy in Pulmonary Tuberculosis. Cochran, J. B., Hislop, J. A. and Clayson, C. *Brit. J. Tuberc. & Dis. Chest*. L: 269, 1956.

The authors report on a series of 19 patients. Clinical, bacteriological and radiological findings are discussed. Doses of cortisone up to 100 mg daily may be safely administered to patients with pulmonary tuberculosis, provided that they are covered with adequate anti-tuberculous chemotherapy. This is true regardless of the stage of the disease. Symptomatic improvement is seen with the combined treatment. The patients have an increased appetite and gain

weight. Fifty percent of the patients will show significant radiological improvement. The hormone may even hasten the resolution of acute lesions. Cortisone did not affect the bacteriology in any way. Patients with negative sputum on direct examination and on culture, did not develop positive sputum during the two month course of therapy. Haematogenous dissemination of tubercle bacilli did not occur. One patient with an indolent radiological infiltrate showed partial clearing on this therapy. A tuberculous lesion was unaffected. Eleven of nineteen patients improved more rapidly

than normal with cortisone. The relatively acute lesions gave the best results.

J. A. LOWDEN

Editor's note: It is interesting to compare the findings in this article with those described in the article by Des Autels et al. The two abstracts are presented together to show the reader that opinions are quite varied on this point. Series of 19 and 15 patients are obviously not large enough to indicate positively the correct answer.

OF CURRENT INTEREST

The Charles H. Best Home for elderly diabetics was opened in Parkgate in the north of England by Dr. Best in July. The other home for diabetics is the one named after Sir Frederick Banting at Kingston-on-Thames, near London.

The Charles Mickle Fellowship for 1956 has been awarded by the University of Toronto to Dr. Arnold Rich, Baxley Professor and Director of the Department of Pathology at Johns Hopkins University and Pathologist-in-Chief of the Johns Hopkins Hospital, for his contributions to the pathology of inflammation, allergy and hypersensitivity.

Dr. William T. Mustard, senior heart surgeon at the Hospital for Sick Children, has been signally honored in England where he addressed, by special invitation, the British Association of Paediatrics. Dr. Mustard chose as his subject: "The Treatment of Complete Anomalous Pulmonary Venous Drainage."

Dr. Donald Fraser of the Hospital for Sick Children has been named 1956 medalist in Medicine, the Royal College of

Physicians and Surgeons of Canada. The medal is awarded annually for the outstanding essay in Medicine and his subject was "Research on Rickets". His essay was presented at the October meetings in Toronto of the College.

Dr. A. W. Farmer has been honoured with the designation QHS—Honorary Surgeon to the Queen, for a two-year period.

Professor R. E. Haist has been elected President of the Canadian Physiological Society.

The following undergraduate awards have been made:

The Phi Delta Epsilon Medical Fraternity Bursary ... P. D. Steinhauer (IV Med.)

A Jean Belton Memorial Bursary

C. W. Barnes (IV Med. Yr.)

Miss D. F. Liedtke (IV Med. Yr.)

G. Roy Sproat and Katherine Sproat Bursaries

Miss S. J. Wallace (IV Med.)

D. E. Andrew (I Med. Yr.)

Scottish Rite Masons' Bursary

R. G. Guest (II Med. Yr.)

Harry Rubinoff Memorial Bursary

J. Schatzker (I Med. Yr.)

FACULTY OF MEDICINE

Promotions in the Faculty of Medicine are as follows:

Dr. Ian Macdonald has been promoted to associate professor of medicine, and director of the newly created Division of Postgraduate Medical Education.

Dr. J. V. Basmajian has been promoted to professor in the department of Anatomy.

Promoted to associate professorships are:

Miss M. T. Wishart (Art as Applied to Medicine)

Dr. A. H. Squires (Medicine)

Dr. J. H. Ebbs (Paediatrics)

Dr. L. N. Silverthorne (Paediatrics)

Dr. W. L. Donohue (Pathology)

Dr. J. W. Scott (Physiology)

Promoted to assistant professorships are:

G. F. Lewis (Anatomy)

J. M. Salter (B. & B. Research)

Dr. J. C. Laidlaw (Medicine)

Dr. R. G. C. Kelly (Ophthalmology)

Dr. J. C. McCulloch (Ophthalmology)

Dr. D. B. French, Dr. G. A. Henry, Dr.

J. B. Whaley (Oto-laryngology)

Dr. J. D. Keith (Paediatrics)

Dr. T. C. Brown (Pathology)

Promoted to associates are:

Dr. H. J. M. Barnett, Dr. B. Berris (Medicine)

Dr. T. C. Jewell (Obst. & Gyn.)

Dr. J. C. Hill (Ophthalmology)

Dr. K. McAskile, Dr. A. M. McLeod, Dr.

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Trichomonads in husbands—Draper says: "The presence of trichomonads in the male genito-urinary tract should always be suspected in the husband of a woman who is repeatedly reinfected."² Karnaky found the parasite in the urethra and prostate or under the prepuce of 38 among 150 husbands with infected wives.³

Carrier cases—Usually the husband re-infects the wife unknowingly. Males harboring trichomonads may be free from symptoms.³⁻⁸

Protection for wives—To prevent re-infection Draper,² Bernstine and Rakoff,⁴ Davis,⁹ and Karnaky¹⁰ advise use of the prophylactic during coitus.

When treating the wife, explain the role of the husband in trichomoniasis and his part in eliminating her infection. To make explanation easier, we have prepared a booklet for your patients, "How THE HUSBAND CAN HELP." Copies on request.

According to the preferences of your patients, prescribe Schmid prophylactics by name. If there is anxiety that a prophylactic might dull sensation, prescribe XXXX (FOUREX)* skins. Made from the cecum of the lamb, they feel like the patient's own skin, are pre-moistened and do not retard sensory effect. If cost

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Treatment for vaginal trichomoniasis—Select a trichomonacide you know will eliminate trichomonads. Data show better than 90 per cent cures with VAGISEC* liquid and jelly, the Davis technique.^{†9} VAGISEC liquid explodes trichomonads within 15 seconds after contact.¹¹

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